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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

CONTENTS:

REVIEW OF ENCEPHALOGRAMS DONE OVER A FIVE YEAR PERIOD 355
Israel H. Schuleman

POLIOMYELITIS IN CALIFORNIA 366
Page Stelle

EFFECT OF L(+) GLUTAMIC ACID ON MENTAL GROWTH 368
Dorothy S. Jaeger-Lee, Emma Gilbert, John A. Washington, Jonathan M. Williams

A NEUROTIC INDEX 375
A. I. Jackman

A CLINICAL REPORT ON THE PALM-CHIN REFLEX 378
John J. McLaughlin and William H. Haines

INDEX 381

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DISEASES of the NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Review of Encephalograms Done Over A Five Year Period

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Galveston, Texas

History

Encephalography is defined by Davidoff and Dyke[12] as the demonstration of the cerebral structures on the roentgenogram after replacement of cerebrospinal fluid with air or some other gas introduced through the lumbar or cisternal route. Dandy[11] was the first to introduce air into the lumbar subarachnoid space as a method of outlining the cerebral structures on the roentgenogram (1919). He apparently obtained the idea for the procedure when some of the ventriculograms he performed revealed filaments on the surfaces of the brain in the sulci. Dandy assumed that air passed from the ventricles, where it had been introduced by the procedure of ventriculography, by normal pathways through which the cerebrospinal fluid circulates and reached the sulci of the brain. Bingel, working in Germany, helped develop this procedure for demonstrating the cerebral structures and called it encephalography (1921).

Technique and Procedure

In the period from January 1944 to January 1949, there were 572 encephalograms performed on 510 patients carried out by one of three services at a university hospital, including neurology, pediatrics and neurosurgery. It would be well to make it clear at this point, that almost every case was carefully screened before the encephalogram was done. This usually meant that the patient had the following examinations as a minimum: 1) history, 2) physical examination including a careful neurological examination, 3) roentgenogram of the skull, 4) lumbar puncture with careful manometric studies and analysis of the chemical and cellular constituents, 5) in many patients, an electroencephalogram was performed. Except for infants and very young children, the procedure was carried out in the X-ray Department. The patient was placed in a chair which was designed and described in a publication by Camp and Waggoner[6] in 1930. It is the general policy of the Neurology and Neurosurgery Departments to drain off all the spinal fluid available and replace it with a slightly smaller volume of a gas. The Pediatrics Department as a general rule, used a smaller amount of gas and did not fully drain the ventricles and subarachnoid space. The examination was done under local or general anesthesia. In either case, a trained anesthetist was present and kept a record of the patient's pulse, respirations and blood pressure. Again, there was an exception in that the Pediatrics Department did this procedure on many infants without an anesthetist in attendance. First year residents in all the above-mentioned departments carried out most of the encephalograms reviewed in this paper. An 18 gauge lumbar puncture needle was utilized. The area to be punctured (interspace L-3 or 4 or L-4 or 5) was surgically prepared and draped. When a successful puncture is obtained, the needle is attached to a syringe with a small piece of rubber tubing and a three-way stopcock intervening. Thus there is accorded flexibility for handling the syringe. A quantity of spinal fluid is allowed to flow into the syringe, but no pressure is exerted by suc-

tion of the syringe. A gas is used to replace the fluid. This procedure is repeated until no more fluid is obtained. It is our policy to put in five to ten cubic centimeters less gas than the total volume of spinal fluid removed. This gradient is built up at various intervals during the operation and not at any one exchange of gas with the fluid. At the termination of the replacement of fluid with a gas, the patient was wheeled into an adjoining room still sitting in the chair and x-rays were taken. Generally an upright posterior-anterior stereoscopic view and either a right or left lateral view was taken. Before further x-rays are carried out, the above exposures are developed and if the pneumoencephalogram is found to be successful by the presence of air in the ventricles, further views are taken. These include:

a) Stereoscopic brow up and brow down with the patient in horizontal position and the x-ray film lateral to the head.
b) Stereoscopic right and left lateral with the patient in a horizontal position and the x-ray film being in the plane of the floor.
c) Stereoscopic view of the so-called reverse occipital exposure with the patient in a horizontal position and the x-ray film being in the plane of the floor.

Thus a total of 13 films are taken on each case. The following physical factors are utilized: a) kilovolts, 65 to 80, b) milliamperes 25 to 30, c) time, 1 to 2 seconds, d) target to Bucky distance 36 inches, e) intensifying screen and f) Bucky which is adjusted for the curve.

Distribution of the Encephalograms

In the five year period reviewed, 572 encephalograms were carried out. A number of patients had multiple encephalograms and in a break down of the figures, we find the following distribution:

1) 440 patients had 1 encephalogram
2) 39 patients had 2 encephalograms
3) 5 patients had 3 encephalograms
4) 2 patients had 4 encephalograms
5) 1 patient had 4 encephalograms and one ventriculogram
6) 2 patients had a ventriculogram and an encephalogram
7) 5 patients had an encephalogram and a ventriculogram
8) 1 patient had two encephalograms and a ventriculogram
9) 6 patients had an encephalogram and an arteriogram
10) 1 patient had an encephalogram, cisternal pneumoencephalogram and a ventriculogram
11) 8 patients had one previous encephalogram at this institution prior to 1944
12) 2 patients had 2 previous encephalograms at this institution prior to 1944
13) 1 patient had 4 encephalograms at this institution prior to 1944

Sex Distribution

There were 183 female or 35.9% and 327 males or 64.1%. In 1000 consecutive cases reviewed by Bohr[1] in 1937, there were 62.8% males and 37.2% females. He suggested that the higher incidence of males was due to their greater exposure to trauma.

Age Distribution

The oldest patient who underwent encephalography was 63 years old and the youngest was 7 weeks of age. Placing the patient in his decade of life, we get the following results:

0-10 years—	95 patients	(18.6%)
10-20 years—	95 patients	(18.6%)
20-30 years—	110 patients	(21.6%)
30-40 years—	93 patients	(18.2%)
40-50 years—	80 patients	(15.9%)
50-60 years—	35 patients	(6.8%)
60-70 years—	2 patients	(0.39%)

In Bohn's[1] series there were between 166-199 patients per decade in the first five decades. He had almost twice as many patients in his report as correspond to this group. It is the general policy of the Neurology Department to restrict encephalography to patients under fifty years old.

Type of Anesthesia Used

The literature is full of discussions about the proper anesthesia to be used in this operation. Patients do have discomfort during and after the termination of this procedure. The majority of patients were given some premedication prior to carrying out the encephalogram. In the adults and older children, this consisted of one of the barbiturates and an injection of codeine. Infants and young children were often done with only a small amount of paraldehyde per rectum. The actual procedure of replacing the spinal fluid with air was done under local or general anesthesia. The general anesthesias used were intravenous sodium pentothal, rectal avertin or nitrous oxide. The following

chart shows the distribution of the anesthesia used in 572 encephalographic procedures done:

a) Intravenous Sodium Thiomal	11
b) Local	118
c) Intravenous Sodium Pentothal	270
d) Avertin	109
e) Nitrous oxide	3
f) Avertin and nitrous oxide	12
g) Children and infants in which no data on the chart about the anesthesia used	33
h) Adults in which no data on the chart about the anesthesia used	16
Total	572

Out of the 118 procedures done under local, 96 (81.3%) proved successful and 22 (19.5%) failed. Out of the 405 done under general anesthesia, 305 (75.4%) were successful and 100 (24.6%) failed. Data on the type of anesthesia used was lacking on 33 infants and young children. However, if we can judge by past performances, these were done under local anesthesia. Twenty-five (78.8%) were successful and 7 (21.2%) failed. Of the 16 adults on which I lack information concerning the anesthesia used, 12 (75%) were successful and 4 (25% failed. General anesthesia was used in 20% of Bohn's[4] reported series. This consisted of ether, nitrous oxide, ether and avertin or avertin and ether. All but nine of the patients were children below 16 years of age. The nine above 16 years of age had generalized tics, low mentality, dystonia or frequent convulsive seizures.

A total of 405 air studies were done under general anesthesia in our series or 77.4%. There was a greater percentage of cases without ventricular fill done under general anesthesia in Bohn's[4] report; 16% done under general failed and 7.4% done with local failed. Davidoff and Dyke[12] reported similar results—theirs being 12.4% failures under general anesthesia and 6% failures under local. My findings generally agree, with 18.7% done under local being failures and 24.6% done under general anesthesia failing. Jungling felt that as a result of the pulsations of the brain which are secondary to the respiratory and heart activity, fluid is forced out of the ventricles into the cisterna magna and air is insufflated in its place. The gas is thought to collect in the cisterna magna around the foramina Magendie and Luschka. When due to the general anesthesia, the intracranial pressure is increased so that pulsations of the brain are decreased, this mechanism may fail. In order to prove this theory, Bohn[4] took eight patients who failed to have ventricular air and gave them intravenous dextrose (25%) to lower the intracranial pressure during a second air injection. Following this, seven of the eight had good filling. The eighth showed some air in the ventricles but had none previously. All the cases had the same anesthesia during the two procedures. As a control, eight patients with unsatisfactory fill had a second encephalogram without dextrose. Of these, only two showed good ventricular fill. Jungling recommended the use of intravenous dextrose in doing a second encephalogram when the first did not fill.

Cleveland[9] reported 75% of his cases done under intravenous pentothal had poor filling of the ventricles and only 20% of those done with local anesthesia revealed poor filling. He repeated 14 of the failures carried out previously under general anesthesia using only local anesthesia and only one of this series did not fill. Von Storch[33] reports that ether and nitrous oxide causes untoward effects upon the intracranial dynamics. However, he advises anesthesia or deep narcosis in carrying out encephalography. Waggoner[34] reported on the use of nitrous oxide and agrees very closely with the above opinion. Solomon[32] used intravenous amytal or pentobarbital or avertin. He felt volatile anesthetics are contraindicated because of 1) pulmonary complications, 2) marked increase in the intracranial pressure, 3) the unpleasant after-effects of the anesthesia. The advantages of the non-volatile anesthesias were: 1) they quickly induce anesthesia, 2) there are no marked unpleasant after-effects, 3) the ease of administration and 4) the prolonged analgesic effect after the anesthesia has worn off. Poppen,[30] Gardner[17] and Nicholson[23] approve of intravenous pentothal. However, there are some disadvantages: 1) trouble getting into the veins, 2) it is contraindicated in children under 7-8 years, as these patients pass quickly through the planes of anesthesia, 3) in hepatic insufficiency, 4) in renal disease, 5) in case of

increased intracranial pressure with signs of medullary compression because of the respiratory depressing effect of pentothal. Levinson[20] recommends the use of avertin in doing air studies on children.

Type of Gas to Replace the Spinal Fluid

Three different gases were used to replace the withdrawn spinal fluid in this group of cases.

1) Air	115
2) Helium	149
3) Oxygen	33
4) Infants and young children in which there was no data about type of gas used	86
5) Adults in which there was no data about type of gas used	189

Of the encephalograms done with air 82 (71.2%) were successful. Those done with helium were successful in 122 (84.6%) cases. Eleven procedures done with oxygen were successful or 33.3%. Of the 86 air studies done on infants and children 69 were successful. Although there was no data on the chart concerning the type of gas that was used to replace the removed spinal fluid, it is the general policy of the Pediatrics Department that did these procedures to use air. Thus there was 80.2% success. Out of the total of 189 encephalograms carried out in which the type of gas used was not known 189 (76.1%) filled adequately. Our over-all success was 76.9%. In 121 cases reported by Frazier,[15] ten per cent showed no filling of the ventricles. He had no explanation for this phenomena.

When gas is injected into the lumbar subarachnoid space, it surrounds the spinal cord and reaches the cranial cavity both ventral and dorsal of the medulla. The gas which is ventrally placed fills the cisternae pontis, interpeduncularis, chiasmatis, lamina terminalis, corporis callosi, ambiens fossae and the cortical subarachnoid spaces. It is felt that this portion of the gas has no part in filling the ventricles. The gas dorsal of the medulla passes through the foramen of Luschka and enters the fourth ventricle, aqueduct of Sylvius, interventricular foramina and finally the lateral ventricles.

Cleveland[11] used helium in performing his encephalograms. He followed this by giving the patient pure oxygen to breathe believing it hastened the escape of the gas from the subarachnoid space and ventricles and alleviates post-encephalographic symptoms. The speed with which the gas passes from the subarachnoid spaces through the vessel's walls and into the blood depends on:

a) Difference in pressures between the gas in the subarachnoid space and the same gas in the blood stream.

b) The inherent diffusibility of the gas itself, which in turn is determined by the size of the molecules of that gas. Light gases are made of small molecules and diffuse more rapidly than heavy gases. Helium diffuses three times as fast as either oxygen or nitrogen. Liberson[22] reports 210 encephalographic procedures using the following gases to replace the fluid: air unfiltrated 40; air filtrated 115; carbon dioxide filtered 3; nitrogen filtered 40; helium 12.

Carbon dioxide dissolved with the spinal fluid and air had to be mixed with it. Reactions were severe using carbon-dioxide and it was felt that it is best to use insoluble gases. Helium seemed to be the most promising of the group.

Poppen[30] used both oxygen and air but reserved the use of oxygen for cases which are done for diagnostic purposes only without the thought of possible therapeutic effects.

Aird[1] worked with anesthetic gases—using them to replace the spinal fluid in dogs. He felt that nitrous oxide and ethylene because of their non-irritative activity, safe sedative effect and good X-ray results might be useful. Ether, divinyl oxide, ethyl chloride and vinyl chloride were considered too dangerous. A later paper[2] reports his results using air, nitrous oxide and ethylene for encephalography in humans. Air absorption was slower than with either of the two anesthetic gases listed above. Ethylene was better than nitrous oxide, as nitrous oxide is absorbed too rapidly for good X-ray pictures. Aird felt that the sedative action of the anesthetic gases tended to lessen the severity of the immediate reaction.

Theories Why Filling Fails

In reviewing the literature on encephalography since the procedure was first brought to attention by Dandy[11] in 1919, I find many theories advanced for the failure of the ven-

tricles to adequately fill. Among those expressed by various authors are:

a) That of Jungling as previously presented.
b) Schott and Eitel attribute it to the difference in the size of the gas bubbles. This depended on the viscosity of the fluid, the rate of injection of the fluid, the size of the needle, the temperature of the gas and the degree of intracranial pressure.
c) Reinberg, in 1925, felt that a meniscus formed because of the difference in the temperature of the gas and the fluid and thus prevented ventricular fill.
d) Heidrich, in 1927, said that flaps of pia-arachnoid cover the foramina of Luschka and Magiendie allowing fluid to escape, but obstructing the inflow of air. Many failures show normal appearance on ventriculograms and may support this idea.
e) Friedman,[16] in 1928, advanced a theory similar to that of Heidrich.
f) Pendergrass,[27] in his publication in 1931, felt that failures were due to an error in technique. He said that the more experienced an operator is, the greater is his success.
g) Bohn[4] reviewed a number of cases in 1937 and felt that increased intracranial pressure, whether it be due to a tumor or the effect of general anesthesia, was significant in accounting for failures with this procedure. Davidoff and Dyke[12] concurred that this mechanism acted in some cases of failure.
h) There is no foramina of Luschka and Magiendie, but drainage of fluid causes the rupture of a thin membrane which in turn allows air to enter the ventricles.
i) Bateman reported on a type of membrane which is not permeable, thus if this type of membrane covers the foramina of Luschka and Magendie, the absence of filling of the ventricles is explained.
j) Absence of filling is due to the collapse of the cortex into the ventricles preventing their filling. Pendergrass says this is not true.

Head Position

In the majority of the encephalographic procedures done in this series, the patient was sitting in a specially designed chair. The head was held by a head rest and was slightly extended. There seems to be little unanimity of opinion about the proper head position or whether it is significant in obtaining a successful encephalogram. Friedman[16] holds the head extended during the injection of air. Fay felt that if the head is flexed on the chest during the period of air introduction and the chin elevated during the drainage period there is more rapid interchange between the fluid and the gas. Poppen[30] suggests that the head be rotated from side to side until no more fluid escapes. Another strong advocate of manipulation of the head in all directions during the procedure is Pendergrass.[29] Adson[1] is against the sitting position in performing this procedure because of the accompanying shock. Davidoff and Dyke[12] believe the upright position of the patient is important in causing the gas to enter the ventricles, but do not believe that manipulation of the head plays a significant role in the filling of these cavities.

Quantity of Fluid Removed with Replacement by a Gas

Davidoff and Dyke[12] wrote in their book on *The Normal Encephalogram* that the question of the optimum quantity of fluid to be removed and the amount of gas to be injected was still in debate. They are strong proponents for the use of small quantities of gas and the removal of equally small quantities of spinal fluid. An attempt to determine the optimum of gas needed in each individual case was made by taking a film in the antero-posterior position after 25cc. of fluid had been removed and 20cc. of gas injected. They felt that only a total of 50 to 70cc. of gas was needed to fill the ventricles and the subarachnoid spaces adequately. Further withdrawal and replacement by a gas resulted merely in evacuating the spinal subarachnoid fluid and replacing it with the gas. Von Storch[33] estimated the total amount of cerebrospinal fluid in the adult to be between 65 and 180cc. These figures were arrived at by doing lumbar encephalograms in adult patients and using those cases whose ventriculosubarachnoid area appeared to be normal size in the X-ray pictures. Solomon[32] felt at least 100-200cc. of air had to be injected to give adequate roentgenograms. He found the patient complained of headache after 30-40cc. of the gas was put in. Pancoast[50] used at least 100cc. air to insure satisfactory results; however, he drained the fluid. Patients had symptoms for an average of 3 to 5 days.

Elsberg[14] found the patients complained of headache after 5-40cc. of air was injected, with an average of 10-20cc. Headaches seemed to occur most often at the time when the injected air had entered the third and

lateral ventricles. Branch,[5] in his series done on children did not drain all the fluid and put 70-90cc. of air in. Levinson's[20] figures coincide with this. Due to the fact that there is an expansion of a gas when it is taken from room temperature of about 80° and put into the spinal canal where the temperature is approximately 98°, Friedman[16] and Pancoast[25] recommended that 10-20cc. more of fluid be removed than gas put in. Carpenter[7] differs from this opinion and feels ten per cent more air may be injected than the fluid removed with safety. He further feels this method gives the best results. It is suggested by Friedman[16] that the operator cease putting gas into the subarachnoid space when gas bubbles appear in the escaping fluid. In such cases, he feels there is an abnormality in the subarachnoid space and continuation is risky owing to the possible herniation of the brain stem into the foramen magnum. The discussion about the amounts of fluid used and replaced by a gas seems related to the possible role the quantity of each substance plays as a causative agent in producing the unpleasant post-encephalographic symptoms. Friedman[16] states that these symptoms are due to the gas and not the removal of the fluid. In opposition to this view, Bohn[4] reports that 32 patients who revealed no ventricular filling differed little from those with good filling of the ventricular system. There have been a number of encephalograms done at the University Hospital in which what should be considered abnormally large quantities of fluid was removed but the X-rays were interpreted as normal. I believe like Pendergrass[28] that the withdrawal of large amounts of cerebrospinal fluid gives further evidence of a pathological condition which may not be apparent on the xrays. The largest exchange in this series consisted of 305cc. of fluid removed and 300cc. of a gas injected. It has been noticed that those patients which revealed cerebral atrophy by encephalography had the mildest post-pneumoencephalographic course. They were often free of symptoms within 24 hours after the termination of the procedure. This is in agreement with Bohn[4] and Davidoff and Dyke[12] who state that the size of the cerebrospinal fluid spaces and the underlying disease of the patient have some influence on the severity and duration of the symptoms. In other words, the duration of the reaction to encephalography depends not on the amount of gas used but is relative to the size of the ventricle. Wolff's[35] recent work on headache mechanisms should be of help in better understanding the cause of this outstanding symptom after an encephalogram. Our patients are placed on a strict regime which insists on a supine position. The patient soon finds out for himself the relief of symptoms relative to this position and their increase on any movements. A rationale for this is found in Wolff's[35] work in which he concluded that with either decreased or increased intracranial pressure, headache results from traction on or displacement of pain-sensitive intracranial structures and is independent of generalized intracranial pressure changes. These pain-sensitive structures are:

1) Great venous sinuses and their venous tributaries from the surface of the brain.
2) The dural arteries and the cerebral arteries at the base of the brain.

Our patients had a morbidity of from 3 to 5 days. I have no statistical data for this impression, however. The figure agrees with that found in many reports.

Changes in the Spinal Fluid Constituents During an Air Study

There is adequate data to support the meningeal reaction that many cases present after an air study. Studies of the spinal fluid after an encephalogram reveal a pleocytosis of 16 to 1933 (Crothers[10]) with 80 to 90 percent neutrophiles. This response is due to the irritating effect of the gases in the ventricules. Penfield[26] reported a white cell count as high as eighteen thousand. In a study made by Levinson[21] in 1939, of changes in the chemistry of the cerebrospinal fluid during encephalography, he found an increase in the quantity of total protein, sugar and phosphatase. These changes he felt, were due to an aseptic meningeal reaction caused by the gas in the ventricles. Since the sugar content is greatest in the ventricles and progressively less in the cisterns and spinal canal, this may explain in part the increase in sugar as the later portions of

fluid withdrawn during encephalography come from the ventricles. The meningeal reaction disappears in 24 to 48 hours. Symptoms lasting longer than this should prompt the attending physician to suspect a septic meningitis.

Apparatus Used to Carry Out the Exchange of Fluid with a Gas

Various methods have been devised during the years to replace the spinal fluid with a gas with the idea of reducing discomfort during and after the completion of the procedure. Dandy's original apparatus consisted simply of a syringe and a needle. I might add he did his encephalograms with the patient in a recumbent position with the head elevated about 20° or more above the lumbar region. Bingel[12] (1922), made use of two lumbar puncture needles. A gas entered one and the spinal fluid was removed by the lower lumbar puncture needle. However, the upper needle had a manometer attached by which they were able to keep track of the pressure. He considered it important to prevent any pressure over 20 mm. of mercury.

Von Storch[33] (1936) believed that the cerebrospinal fluid could not be replaced by gas through a single aperture without causing alternate depression and elevation of the subarachnoid and intraventricular pressures. These shifts of pressure vary directly with the volumes replacing each other. He felt these alterations contribute to the discomfort and untoward reactions of the patient. These variations are thought to produce alternate constriction and dilation of the cerebrovascular bed and massages the intracranial contents. It is further suggested that the pressure shifts may be minimized by the exchange of small volumes. Davidoff and Dyke[12] are in accord with the idea of using small exchanges between the gas and the spinal fluid, but feel it can be done with only a simple apparatus consisting of a syringe, one lumbar puncture needle, a three-way stop cock and a small piece of rubber tubing. The patients in this survey had their procedures done in a manner similar to that described by Davidoff and Dyke.[12]

Findings on the Encephalograms in this Review

Out of the total of 510 patients in which encephalography was performed, 232 (45.5%) of the cases were reported to be abnormal. Except in a few cases where the clinical staff disagreed the report of the X-Ray Department was accepted. In these cases, the latter opinion was taken. There were 34 (6.7%) definitely proven tumors, and 8 (1.6%) questionable spaceoccupying lesions found.

The term space-occupying lesions and tumors is used to mean one of the following: a) neoplasm, b) subdural hematoma, c) abscess, d) aneurysm, e) bony tumor of the skull. This mass of patients is a rather heterogeneous group as far as their presenting symptoms are concerned. For the purpose of obtaining a better understanding of any reasons for our success or failures in the procedure of encephalography, I have divided the cases according to the following predominant complaints:

a)	Convulsive disorder	355 patients
b)	Headache	56 patients
c)	Organic brain syndrome	24 patients
d)	Paralysis	29 patients
e)	Retarded	23 patients
f)	Visual disturbance	17 patients
g)	Miscellaneous	6 patients

1. Torsion spasm
2. Skull defect
3. Diabetes insipidus

Of necessity there is a great overlap in such an arbitrary plan. If we further break down the total number of tumors found according to the predominant complaint, we find the following results:

	Abnormal Encephalograms	% of total abnormal encephalograms	Tumor found	% of patients with tumor	? Tumor found	% of patients with tumor	Tumor removed previous to encephalogram
	No.		No.		No.		
Convulsive disorder	158	67.8%	20	5.6%	3	0.8%	10
Headache	18	7.8%	7	12.5%	1	1.8%	2
Organic brain syndrome	16	6.9%	3	12.5%	0	0	0
Paralysis	18	7.8%	0	0	0	0	0

Retarded	15	6.4%	0	0	1	4.3%	0
Visual disturbance	6	2.6%	4	23.5%	3	17.7%	1
Miscellaneous	1	0.43%	0	0	0	0	0
Total	232						

Out of 34 patients with a definite tumor, (13 more had been removed before this study) five who had one encephalogram failed to successfully fill and to reveal a space-occupying lesion. The presence of a tumor was later proven by other methods (ventriculograms, operations, etc.) The histology of these lesions were reported as:

Glioma	1
Ependymoma	1
Pinealoma	1
Pituitary adenoma	1
Oligodendroglioma	1

Six patients who had normal encephalograms, were later proven to have a tumor:

1. Angioma	1
2. Aneurysm	2
3. Craniopharyngioma	1
4. Osteofibroma of skull	1
5. Glioblastoma	1

One glioblastoma case had two pneumoencephalograms of which the first procedure failed to fill. A patient with a right thalamic lesion not operated on but proven by a ventriculogram, had two encephalograms with both failing to fill. Four encephalograms were carried out before the last one was successful and revealed a space-occupying lesion (A.V. angiomata) in one patient. One patient who was later shown to have an astrocytoma had four encephalograms. All but one revealed no air in the ventricles and the fourth was interpreted as normal. One case had two air studies in which both were interpreted as bilateral cortical atrophy and on an operation performed later, an astrocytoma was removed. A subdural hematoma was revealed in one case after a ventriculogram was interpreted as normal. In eight cases reported as a questionable tumor, seven failed to show air in the ventricles. The locations of the lesions were:

a) posterior fossa	1
b) mid-brain	2
c) glioma of the optic chasm	1
d) tumor (location?)	3

An eighth questinable finding thought to be a callosal tumor had a successful encephalographic procedure.

hus, only 21 patients of the 42 with space-occupying lesions had such tumors proven by encephalography. hree of the cases had two or more air studies carried out:

a) A. V. angioma	3 failed and 1 successful
b) Glioma	1 failed and 1 successful
c) Subdural	ventriculogram failed and pneumoencephalogram successful

The following chart will summarize the general finding in this five-year series of patients.

Encephalogram & Type of Lesion	No.	%
NORMAL:		
Tumors	7	(1.4%)
No lesions	197	(38.6%)
ABNORMAL:		
Cortical atrophy and arachnoiditis	142	(25.9%)
Space-occupying lesion	21	(4.1%)
Porencephaly	5	(00.98%)
Cerebellar atrophy	2	(00.39%)
Agenesis corpus callosum	1	(00.19%)
Cortical atrophy (later found tumor)	1	(00.19%)
? Corpus callosal tumor	1	(00.19%)
UNSUCCESSFUL:		
Later found to have tumor	6	(1.1%)
? Tumor	7	(1.4%)
All but tumors	120	(23.5%)
TOTAL	510	

The patients with tumors who had normal air studies had the following types of lesions:

Aneurysm	2
Craniopharyngioma	1
Angioma	1
Glioblastoma multiforme	1
Osteofibroma	1
Astrocytoma	1

In Bohn's[4] series of 1,000 cases which had encephalograms, 10 per cent had brain tumors. However, 26 per cent of the failure had neoplasms. He reports the following distribution among his cases:

Normal	338
Cerebral atrophy	342
Tumor	98
Porencephaly	31
Unsatisfactory fill of the ventricles	83
(10 later shown to have a tumor)	

It is often said that encephalography is of little help in diagnosing the neurological dis-

eases of children. Crothers[10] (1930) believes they are of positive value. His series of 200 cases revealed evidence of cerebral atrophy in about 50 per cent. Casamajor[9] (1949) working with children, found that in 21 per cent of the cases with a definite neoplasm existing, the encephalogram did not reveal the tumor.

Complications of the Procedure

Except for the reactions to encephalography already discussed and a rare fatality, Davidoff and Dyke[12] say there are few complications encountered to this procedure. Kerman[18] (1943) reported a case of bacillus pyocyaneus meningitis in a child following encephalography. The authors cultured the same organism from the wash bottle through which the helium gas was bubbled for the procedure. No similar occurrence is reported by Davidoff and Dyke[12] in over 8000 cases. Kerman[18] also reported that the literature had reports of 20 cases of pyocyaneys meningitis following lumbar puncture. Penfield[26] (1925) reported a case of staphylococcus albus meningitis which was proven by culture in a series of 60 procedures. The one death reported by Friedman[16] out of 42 encephalographic procedures occurred 48 hours after the termination of the procedure. The patient had meningeal signs, but no organisms were cultured. In this series, a five month old infant died on the sixth post-encephalographic day. The patient had an elevation in temperature coming on a few days after the procedure was terminated until his death. The infant had diarrhea following the air study. He had been brought to the hospital because of convulsive seizures. The encephalogram was successful and interpreted as revealing generally dilated ventricles. Necropsy was carried out and the final diagnosis was: 1) enterocolitis, 2) subacute meningitis, 3) malformation of the brain—cytoarchitectural type. No data was found on the chart about bacteriological studies either pre or post-mortem.

A 43 year old man with a history of convulsive disorder had a sudden onset of left hemiparesis four days after encephalography. The patient had a negative neurological examination previous to the procedure. The air study was interpreted as revealing some inequality of the ventricles—left larger than right. The patient had a slow but never complete return of function of the paralyzed side. He had been up and about the ward for two days before the onset of his accident. There was a transient episode of expressive aphasia occurring in a forty-one year old man who had a convulsive disorder. The encephalogram revealed only bilateral enlarged subarachnoid channels. Rosenbeck[31] reports a case of right hemiplegia with aphasia coming on the next morning after encephalography in a 45 year old man. The patient had been seen for headache and was neurologically negative. He felt this complication was probably on a vascular basis and discussed the following possible mechanisms for such an accident:

1) Air emboli not a satisfactory explanation because of the barrier between the blood vessels and the subarachnoid space. King[19] and Ostenasek reported two cases of death from air embolism during encephalography. They pointed out that the most likely site for air to enter the venous system would be a tear of a small vessel of one of the dural sinuses.
2) The removal of ventricular fluid might remove a certain amount of support to the adjacent blood vessels. Against this possibility is the large number of spinal punctures without such mishap.
3) The mechanics of circulation in the blood vessels adjacent to the lateral ventricle is disturbed. The alternate rapid lowering and elevation of the blood pressure in sclerotic vessels is apparently too much for their weak walls and ruptures may occur.

Dixon[13] (1932) lists the presence of arteriosclerosis as detectable in the optic discs as a major contraindication to encephalography. By not doing air studies on patients over fifty years of age, we hope to prevent such occurrences.

There are a number of deaths after encephalography reported in the literature. An exact estimate of the mortality rate is difficult because the criteria by which a death is considered related or not related to the encephalographic procedure varies. I believe it may be significant too, that in the early stages of this procedure many people with cerebral tumors had such air studies—today this is considered hazardous. In Pancost's[24] report, he has compiled a total of 1529 encephalographic procedures done by 17 different clinics.

Age	*Sex*	*Chief Complaint*	*Neurological Findings*	*Complications*	*Time of Onset*	*Result of Encephalogram*	*Pathology*
43	M.	C.D.	Negative	Left hemiparesis	4 days	Left ventricle larger than right	—
23	M.	C.D.	Negative	Severe shock on two occasions	Immediately	Unsuccessful—two occasions	Right thalamic tumor
35	F.	C.D.	Weakness, right extremities	Severe shock	Immediately	Normal	—
41	M.	C.D.	Negative	Expressive aphasia transient	2 days	Increased subarachnoid channels	—
48	M.	C.D.	Negative	Signs of increased intracranial pressure	Immediately	Shift of ventricles	Astrocytoma
6	M.	C.D.	Negative	Lumbar puncture, needle broke. Removed by neurosurgery	During procedure	Unsuccessful	—
21	M.	C.D.	Negative	Convulsion	During procedure	Unsuccessful	—
10	F.	C.D.	Negative	Bloody tap	During procedure	Unsuccessful	—
7	M.	C.D.	Negative	Air would not go in (probably poor tap)	During procedure	Unsuccessful (2nd one normal)	—
24	M.	C.D.	Left hemiparesis	Fluid became bloody after 77cc. removed	During procedure	Substandard	—
39	M.	C.D.	Negative	All taps bloody	During procedure	Unsuccessful	—
52	F.	C.D.	Negative	Dyspnea and area infiltration of right chest. X-ray ? pulmonary embolus	2 hours	Normal	—
26	F.	Headaches	Negative	Unable to void for 3 days	After procedure	Unsuccessful	—
49	F.	Headaches	Aphasia	Severe shock (patient had hypertension)	During procedure	Unsuccessful	Thrombosis left middle cerebral artery (arteriogram)
52	F.	Organic brain syndrome	Negative	Respiratory and circulatory embarrassment. Confused, etc.	After procedure	Unsuccessful	Ependymoma (died 20 days later)
15	F.	Visual loss	Optic atrophy O.U.	Elevated temperature and respiratory embarrassment	Few hours	Unsuccessful	Pinealoma
5mos.	M.	C.D.	Negative	Meningeal signs	Few hours	Successful	a. subacute meningitis b. brain malformation (architectural type)

*C.D.—Convulsive disorder

Davidoff and Dyke[12] reported 9 deaths, 0.22 percent, in their original series of 4,000 cases of encephalography. Of the 9 deaths, 2 patients had aneurysms, and 4 had brain tumor. Of the other 3, one was a child with advanced central sclerosis; the second was a child with unknown cerebral disease who suffered a subarachnoid hemorrhage. The final case was a man who had a normal encephalogram but died from a coronary occlusion six hours after the procedure. Two deaths occurred in Bohn's[4] of 1000 cases. Both had verified brain tumors.

Excluding the usual reactions patients have during and after encephalography, there was a total of 18 complications occurring in our 572 encephalographic procedures. Three deaths occurred. The first was in a 15 year old girl with the chief complaint of progressive visual loss and on examination there was bilateral optic atrophy. The ventricles failed to fill. The evening of the procedure her temperature was 106° rectally. The patient was operated on at which time a pinealoma was found in the region of the sella. The patient died five days after the encephalographic procedure. The second death occurred in a five month old infant and has been described completely above in another section. The third death occurred in 52 year old woman. The air study was unsuccessful and the patient appeared worse after the procedure. This was manifested by circulatory and respiratory embarrassment. The patient died 20 days after the termination of the procedure. Necropsy revealed an ependymoma. A complete summary of the complications is contained found in the following chart:

Summary

1. The encephalograms done at a University Hospital from January 1944 to January 1949 are reviewed. This is comprised of 572 air studies on 510 patients.

2. Out of 405 encephalographic procedures done under general anesthesia, 305 or 75.47% were successful; 118 air studies were done with local anesthesia, with 96 or 81.3% proving successful.

3. When air was used to replace the spinal fluid there was a success of 71.2%; with helium, 84.6% fully filled and with oxygen, only 33.3% had a successful procedure.

4. A tumor was proven in 34 or 6.7% of this series and in 8 or 1.6%, a questionable space-occupying lesion was found. Seven patients with normal pneumoencephalograms were later shown to have tumors.

5. Eighteen patients had reactions felt to be a complication of the procedure. Three deaths are reported, but it is difficult to specifically relate them conclusively to the air study.

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Poliomyelitis in California

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During the thirty year period, 1915 to 1946, a total of 18,411 cases of poliomyelitis have been reported in California. Only three states, Minnesota, Vermont, and New York have had higher case rates per 100,000 population, for this period.

Gilliam, Hemphill, Ference and Dauer have emphasized the significance of the incidence variations in counties. The purpose of this study is to determine the county rates of incidence in California, and to ascertain, if possible, reasons for the differences. To date, seven states have been surveyed in this manner, results warranting a continuance of this work.

Table 1 is a list of the 58 counties of California, the average population, the number of cases reported in each county from 1915 to 1946, and the case rate per thousand population.

TABLE 1

County	Average Population	No. of Cases	Rate per Thousand
1. Calaveras	7,568	10	1.25
2. Mariposa	4,174	6	1 5
3. Plumas	9,635	18	1.87
4. Butte	51,437	110	2.16
5. San Benito	11,507	25	2.2
6. Lassen	13,495	31	2.4
7. Napa	29,613	74	2.45
8. Imperial	56,669	141	2.49
9. Placer	28,121	70	2.49
10. Trinity	3,594	9	2.5
11. San Francisco	634,090	1,669	2.63
12. Mendocino	29,020	77	2.7
13. Modoc	7,955	22	2.75
14. Tuolumne	10,111	28	2.8
15. Monterey	71,157	206	2.89
16. Amador	8,590	25	2.91
17. Siskiyou	25,785	78	3.03
18. San Mateo	115,012	352	3.06
19. Alameda	516,517	1,655	3.2
20. Colusa	10,226	34	3.23

21. San Bernardino	162,165	526	3.25
22. Sacramento	169,780	553	3.27
23. El Dorado	11,002	37	3.36
24. Ventura	66,700	228	3.42
25. Santa Clara	77,420	596	3.44
26. San Joaquin	129,397	455	3.44
27. Mono	1,675	6	3.5
28. Santa Cruz	43,670	158	3.6
29. Shasta	23,038	84	3.7
30. Alpine	261	1	3.83
31. Merced	44,428	171	3.85
32. Sutter	17,387	69	3.86
33. Sonoma	71,534	287	4.01
34. Orange	131,217	533	4.06
35. Lake	8,002	33	4.12
36. Glenn	12,581	52	4.13
37. Sierra	2,398	10	4.17
38. Solano	58,326	246	4.22
39. San Diego	286,805	1,212	4.23
40. Humboldt	48,752	210	4.3
41. Riverside	101,559	438	4.31
42. Marin	51,783	226	4.36
43. Nevada	15,035	66	4.44
44. Madera	22,361	101	4.51
45. Santa Barbara	68,640	311	4.53
46. Contra Costa	132,607	604	4.56
47. Tulare	98,084	451	4.59
48. Kings	32,212	151	4.69
49. Stanislaus	75,457	355	4.7
50. Inyo	8,024	38	4.75
51. Yolo	27,111	129	4.77
52. Fresno	181,487	883	4.87
53. Yuba	15,748	83	5.29
54. Los Angeles	2,513,938	13,568	5.4
55. Del Norte	5,092	28	5.7
56. Tehama	15,058	91	6.0
57. San Luis Obispo	33,967	207	6.1
58. Kern	124,925	1,354	10.83

From this table, 29 identical graphs were prepared, showing the incidence rate for each county, the curves starting with the lowest county on the left hand side, to the county of highest incidence on the right. Then another curve was drawn in on each graph, each representing some county feature of climate, geography or agriculture. If any connection or relationship between feature and polio existed, the second curve should parallel or match the incidence curve. Figures for the climate of counties were taken from "California Blue Book." Figures on agriculture were taken from U.S. Census, "Preliminary 1950 Census of Agriculture."

TABLE 2

County population	x	
County area		0
County rainfall, annual		0
County temperature		0
Number of farms each county	x	
Percent of land in farms		0
Number acres irrigated	x	
Oats, acres	x	
Potatoes, acres	x	
Barley, acres	x	
Number of milk cows	x	
Number of cattle, calves	x	
Number of hogs	x	
Number of turkeys	x	
Number of horses	x	
Hay, acres	x	
Number of sheep		0
Number of chickens	x	
Corn, acres	x	
Wheat, acres		0
Beans, acres		0
Vegetables, acres		0
Apples, acres		0
Peaches, acres		0
Pears, acres		0
Plums, acres		0
Prunes, acres		0
Apricots, acres		0
Grapes, acres		0

(Mean, June to October)

The curves of features identified by an x, parallel the incidence curve. The features followed by a zero, show no relationship to polio. It will be noted that all farm animals, except sheep are in the x classification. All farm animals together with sheep contribute to the breeding of flies, but sheep may lack the public health significance of other farm animals, being often pastured miles from human habitation.

Fruit has often been suspected as playing some part in the spread of poliomyelitis, because its appearance in markets and on the table is, in many localities, coincident with the marked seasonal increase in case numbers, in August. The selective preferences of certain insect species for one kind of fruit has apparently no significance in the poliomyelitis of California.

Effect of L(+) Glutamic Acid on Mental Growth

(A Study of 51 Cases)

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This study concerning the long range effects intellectually and physically of glutamic acid on mentally retarded children was initiated in 1946 following favorable reports by Waelsch[1,2] and Zimmerman.[3] Considerable controversy has since then centered around the effectiveness of glutamic acid administration. Our experience is offered as a basis for examining critically many of the issues involved, and the design of the experiment was geared to avoid pitfalls encountered by others.

The patients were obtained from three sources. The majority came from the Child Welfare Clinic, and many of these had been studied and recognized as mental defectives with observations extending over many years. The second source was physician referrals of private patients and the third was direct application by the parents of mentally defective children. All were treated on an out-patient basis. Children taken in the project were integrated in sequence of their applications and control over glutamic acid therapy was solely in the hands of the physicians involved in the research project. There were 101 children ranging in age from a few weeks to 14 years, with an I.Q. ranging from 30 to 100. For purpose of analysis they were divided into two groups: those testable with the 1937 Revision of the Stanford-Binet (mental age two years or more), and those not testable by this method. The psychological data to be reported in this paper refer only to the first group which represents 51 per cent of the total number.

The children came from all income levels. The majority had no neurologic manifestations, nor was there a history of mental deficiency in the family. The children cannot be considered homogeneous for the term "simple mental retardation" represents a catch-all for all mental defectives who are unclassified by present means. In view of this fact the problem of adequate control of experimental observation was acute and was attacked in two ways. The first was the method that has been used by Zimmermann and by Sister Maureen Harney,[4] namely, the use of the previous mental growth of the patient for comparison with any change on glutamic acid. Another method was to compare the treatment period with the changes occurring when treatment was stopped, and also the comparison of the treatment period with a final period of placebo.

The children underwent the following work-up before being admitted to the project: a history with emphasis on prenatal diseases in the mother, birth accidents, and neonatal development was obtained; a general physical examination with evaluation of general physical status was made by a pediatrician; x-rays were taken of the hands to determine whether the number of epiphyses was within the normal range for the age; the weight and height of all children were plotted according to the method of Wetzel, and information for many years previously was obtained by contacting school health departments and physicians, and incorporated in the Wetzel Grid; a psychological examination was made with the basic test being the Binet for all children of mental age two years or over; the Gesell Development Schedule was given all children with mental age less than two years; many other tests, including Cattell, Rorschach, and Goodenough, were given to those children. The test values reported in this paper were all obtained by the same psychologist (E.G.) except where otherwise noted. This report

From the Research Foundation of Children's Hospital, Washington, D. C.

deals only with values obtained using forms L and M of the 1937 Revision of the Stanford-Binet.

A neurological examination was done on each child by a neurologist, who ordered such examinations as electroencephalograms and pneumoencephalograms as were indicated. Blood specimens were taken for a blood count, hemoglobin, cholesterol, serum protein, thymol turbidity, cephalin flocculation and glutamine levels. A routine urinalysis, with inclusion of a specific test for phenylpyruvic acid was made. Ten children were given basal metabolism tests before and after single doses of glutamic acid.

After the initial work-up the patients were given powdered glutamic acid to tolerance. The instructions to the mother were to give the patients ½ teaspoon of the powdered material (2 grams) 3 times a day, mixed with whatever food would be most compatible, and to increase this dose until the child developed symptoms of toxicity, such as irritability, sleeplessness, nausea and vomiting. The dose was then to be diminished. In many children the dose could not be increased to toxic levels, for they refused to take more than moderate amounts of this distasteful material.

At the end of varying intervals, ranging up to 2 years, glutamic acid was stopped and no treatment, or lactose placebo in equal quantity, was instituted. The children were followed by serial retests every six months on alternating forms L and M of the Binet, and repeat physical and blood examinations were done at the same time. The procedure was so organized that in all cases the psychological examination preceded the taking of blood specimens in order to maintain as good a rapport with the child as possible. During the course of the two years with which this report is concerned, 25 cases discontinued glutamic acid therapy. Of these 19 discontinued therapy with glutamic acid voluntarily, because they refused to take any more of the material. In order to test the effect of a placebo as compared to previous glutamic acid therapy, all cases which came up for testing in the last ten months of the study were switched to placebo without knowledge of the parents.

During the entire study close contact was maintained both by telephone and by personal interview with the parents. Case records were kept of any changes that might have occurred in the home situation; in parent-child relations; and in the intake of glutamic acid. Parents came to the clinic every 3 or 4 weeks to receive additional supplies of glutamic acid and were questioned as to their impression of the child's progress.

TABLE 1

DISTRIBUTION OF INITIAL POPULATION

	Mean *Mean* ± *1 S.D.** *Mean* ∓ *1 S.D.**	*Mean* ∓ *2 S.D.** *Mean* ± *2 S.D.**
IQ. 64.59 ± 14.95	68.5%	96.4%
Age 7.85 ± 2.72 yrs.	65.4%	98.1%
Theoretical Distribution for a Homogeneous Population	68.3%	95.5%

* S.D. = Standard Deviation.

RESULTS

The initial I.Q. of our group of patients was 64.59 ± 14.95 range 37—99, age 7.85 ± 2.72 years. As can be seen from Table 1, the distribution of our patients with respect to I.Q.'s and age was homogeneous and in conformity with the standard curve. Table 2 gives the numerical values for I.Q. change over the intervals 6, 12, 18 and 24 months, and Table 3 gives the change of I.Q. 6 months after discontinuance of treatment or substitution of placebo. Since a probability of chance deviation less than .01 (one chance in a hundred) is considered highly significant, it can be seen that the mean increase in I.Q. achieved by these patients is far beyond the realm of chance. The change was calculated on the basis of the last test before cessation of glutamic acid treatment and the first test 6 months afterwards. This last group includes children treated for from 6 to 24 months with an average of about 18 months.

TABLE 2

CHANGE IN MEAN I.Q. AFTER GLUTAMIC ACID TREATMENT

51 Children—Mean Initial I.Q. 64.59 14.95

Period of Treatment	Number	Mean Change	S.D.	Probability
6 Mos.	51	+ 7.37	6.29	<.001
12 Mos.	42	+ 9.02	7.16	<.001
18 Mos.	28	+11.82	9.76	<.001
24 Mos.	13	+13.77	12.18	<.01

TABLE 3

CHANGE IN I.Q. SIX MONTHS AFTER DISCONTINUANCE OF GLUTAMIC ACID

	No.	Change	S.D.	Probability
1. Compared to Last Test on Glutamic Acid	25	—6.68	5.79	<.001
2. Compared on Initial I.Q.	25	+3.20	6.86	<.05
3. Patients on Placebo 6 Mos.	6	—4.50	6.16	<.05
4. Patients With No Treatment for 6 Mos.	19	—7.36	5.69	<.1

The question of the effect of placebo versus no therapy at all was investigated and the results are summarized in Table 3. Although there seems to be less of a drop on placebo than on no therapy at all, it is probably a chance variation since there is no statistical difference between this value (P> .05) and the value for the whole group. The question of the effect of placebo on the mother-child relation as it may alter the I.Q. was considered in this analysis and is being studied more extensively. So far no significant effect can be demonstrated. On the other hand, the comparison of the mean I.Q. 6 months after the cessation of therapy, with the original I.Q. shows a residual gain

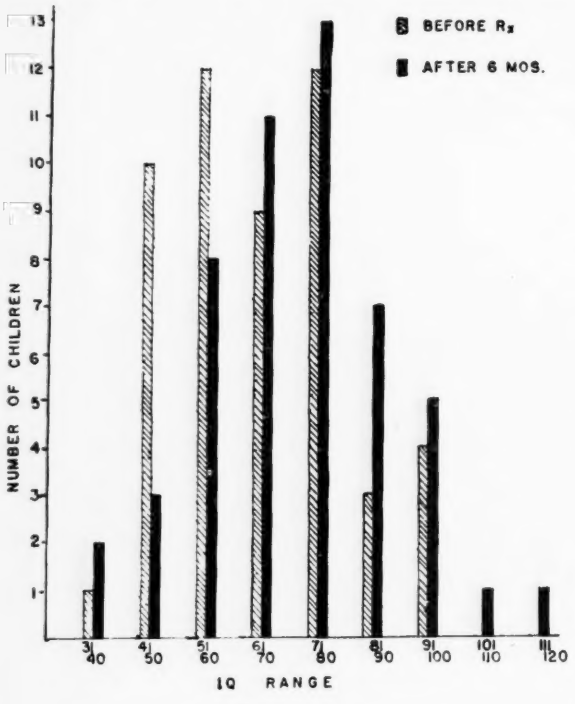

Figure 1.

of 3.72 points, which is statistically significant. That the change in I.Q. occurred relatively homogeneously throughout the group can be seen from Fig. 1, which shows the distribution of I.Q. groups before treatment and after 6 months of glutamic acid therapy.

The rate of mental growth on treatment with glutamic acid differed markedly from the previous rate of growth of these children. There were three or more tests before treatment for 15 children of this group. These data are summarized in Fig. 2. Each curve represents the mental growth history in terms of mental age of a single child. The mean I.Q. of this group at the initiation of therapy was 63.0, and the mean 6 month gain was 8.75 points. These figures correspond to the mean I.Q. and gain of the entire group of 51 patients. (Table 1.) The data were plotted by taking the mental age at the time of institution of therapy as the zero point and plotting all mental ages obtained before and after this point as differences from the base value. A family of curves was obtained which, when treated statistically, can be resolved into two separate straight lines, one leading up to the base point, representing the weighted mean change in mental age with time for the whole group up to the initiation of therapy; the second line, leading up from the base point, representing mental growth on glutamic acid treatment. The highly significant difference (T=4.23, P<.001) between the two mean slopes shows that the mental growth of the children treated with glutamic acid was greater than what the previous growth history for the child would predict. The mean rate of mental growth on therapy was three times the original rate. Since the mean I.Q. and the 6 month gain in treatment correspond so closely with the figures for the entire group of 51 patients on whom we had extensive previous data, a fair picture of the behavior of the whole group is obtained.

The dosage of glutamic acid has been compared to the amount of change in I.Q. in Fig. 3. There is only slight correlation of I.Q. change with dosage. The maximum change in I.Q. during therapy is plotted against number of children in Fig. 4, and demonstrates that the mean changes of Table 2 were not based on marked increases for only a few of the group. More than half the group showed increase in I.Q. at some

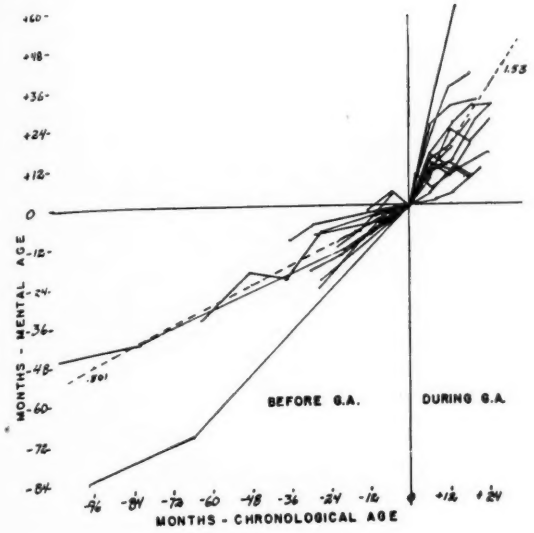

Figure 2.

time on treatment of greater than 11 points. The relation of improvement in I.Q. was plotted against chronological age, mental age and original I.Q., but no correlation was demonstrable.

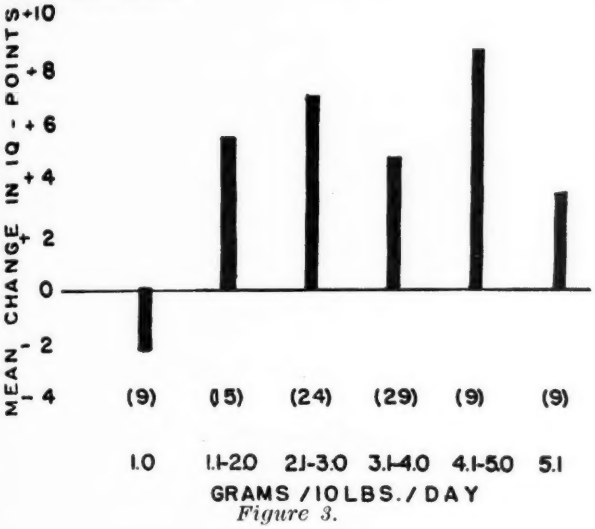

Figure 3.

DISCUSSION

There have been a number of reports concerning the effect of glutamic acid on mental deficiency in children. On the positive side are the reports of Waelsch,[1,2,9,10] Zimmermann,[3,11] Sister Maureen Harney,[4] and our group, and on the negative side are the reports of McCulloch,[5] Loeb and Tuddenham,[6] Ellson, Fuller and Urmston,[7] and Kerr and Szurek.[8] Differences can be detected among these reports, which suggest that the investigators were studying cases under different conditions. All of the negative reports deal with institutionalized patients, whereas all the positive reports relate to out-patients, except for that of Sister Maureen Harney. Her group of children, however, was selected on the basis of I.Q. for attendance at a special school for mentally retarded children. Thus, even this group would be more closely related to the group showing improvement than to the institutionalized mental defectives studied by the investigators who could demonstrate no effect of glutamic acid therapy.

The average I.Q. for the children in the groups having favorable response was more than 10 points higher than the I.Q. for the negative groups. Furthermore, the type of environment in which improvement is to be expected to occur probably influences any results obtainable with therapy. This point is mentioned in the report of Ellson et al. in explaining their finding that their control group improved as well as did their treated groups on glutamic acid, "consequently, the stereotyped institutional life of the subjects was very probably livened up by extra attention. The subjects in both groups also made two trips a day to the school kitchen. This additional stimulation may have produced some effect on test scores." Since the I.Q. is influenced by the "cultural" pattern surrounding the child, it might be expected that variations in I.Q. produced in patients

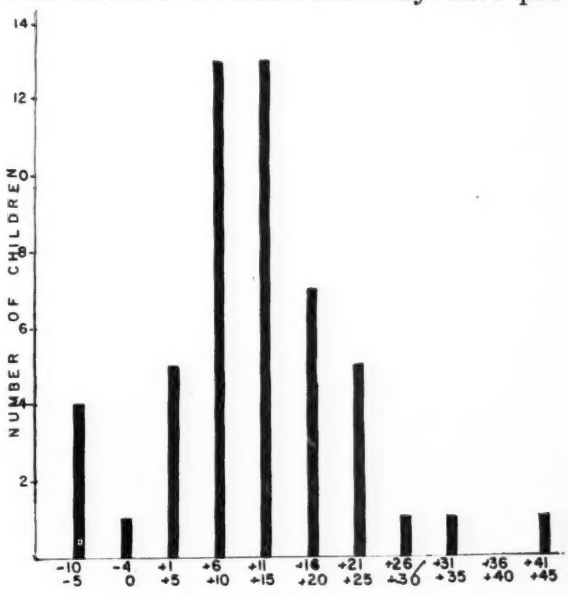

Figure 4.

residing in a family atmosphere would be different from those obtained in an institution. Material relating to this problem is discussed by Sarason.[13]

A major criticism of the work of Zimmerman has been that the tester was aware that the patients were under treatment, and that this would tend to influence test scores.[5] In our investigations the same situation was present, namely, that the psychologist was aware that the patients were under treatment. Therefore, a study was made of the correlation between the test results obtained by our psychologist and the data obtained on the same patients by other psychologists who were unaware of our test results. These tests were done by psychologists in other institutions for various purposes, such as placement in school, evaluation by social service agencies, and medical consultations. Each patient is designated in Fig. 5 by a symbol appropriate to the situation of therapy under which he was tested by both psychologists. If there were bias in any significant degree, one would expect the following results: The majority of the tests by our psychologist before treatment with glutamic acid should be lower than, or equal to, values obtained by other psychologists (below the diagonal which represents perfect agreement). When the patient is on therapy, our psychologist should report tests higher than the values obtained by other psychologists (above the diagonal). The values obtained by our psychologist with the patient on placebo, or on no therapy, should be lower than, or equal to, those obtained by the other psychologists. Thus, if there were bias, the distribution of points on the curve would show the majority of the three groups, before therapy, on placebo, and on no therapy, falling on or below the diagonal lines, whereas the majority of the tests on glutamic acid would fall above the line. As can be seen from Fig. 5, this is not the case, for there is equal distribution of points in all groups above and below the line. The correlation coefficient of our psychologists' data with that of the other psychologists is .923, which agrees closely with the expected test-retest reliability of the Stanford-Binet. All comparative tests depicted on the chart are based on intervals of 11 months or less; all of the points for periods on treatment or after, are for less than 6 months.

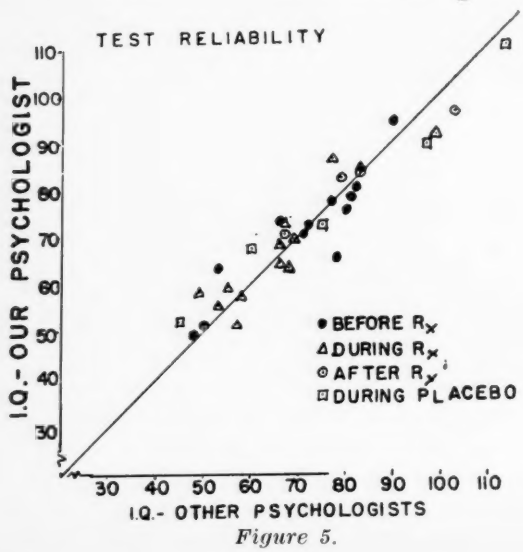

Figure 5.

A second factor suggested in many criticisms of the use of follow-up tests is the amount of "practice effect." The data obtained prior to treatment with glutamic acid have been analyzed for this point. For 19 patients over an interval of 3 to 8 months, average 4.7 months, tested by several testers, the mean initial I.Q. was 66.58 ± 13.00. The I.Q. 4.7 months later was 66.74 ± 13.84. This indicates no significant difference in test-retest values. For 6 patients tested in an interval shorter than 3 months, the mean difference was +1.3. These results are in accord with a considerable body of psychological literature which indicates that the shorter the interval between tests the greater the residual learning,[14] and the results over the longer interval confirm the evidence that residual learning, particularly in mental defectives, does not hold up over a long period of time.

The report of Loeb and Tuddenham[6] concerns a group of patients, at least half of whom were older than the patients studied in our group. Furthermore, the basis for comparison used was the mental age instead of the intelligence quotient. The use of mental age in such a manner, although defended by Loeb and Tuddenham on the basis of the "artificiality" of the I.Q. score, conflicts with the observations of Wechsler, who pointed out that it is impossible to compare a mental age of an adult with that of a child. "The basic reason a mental age of

12 at 12 does not mean the same thing as the mental age of 12 at 30 is that the measured ability, and hence the mental age scores, represent different portions of the subjects respective total intelligence."[15] The extreme variations of mental age found by these authors suggest that factors such as patient interest, recalcitrance, and rapport were also operating, particularly in the older patients.

Another complication in the use of older groups of patients for study of the effect of glutamic acid on mental function is the diminution of rate of learning with age. As described by Wechsler, this diminution in rate of increase begins to occur in normal people around the age of 12, and he cites evidence indicating that this decline begins even sooner in mental defectives.[16] A therapeutic agent aimed at increasing mental growth might well be expected to be most effective during the period when mental growth is at its highest and least effective when such growth is on the decline.

The study reported by McCulloch[5] is different from those studies in which positive results were obtained on the basis of two characteristics of his group of patients. First, their mean I.Q. was considerably lower —twenty points—than that of the groups which had success with glutamic acid. Second, the type of mental defectives studied included slightly less than 50 per cent Mongoloids and about 30 per cent familial mental defectives. It is apparent that from diagnostic criteria, the populations dealt with in the studies which showed an effect of glutamic acid differ from this population. As seen from Table 4, our group was composed chiefly of simple mental defectives and included no Mongoloids. The only familial defective patient that we had showed no improvement on glutamic acid.

TABLE 4

I.Q. CHANGE BY DIAGNOSIS

Class	No.	6 Mos. Change	No.	12 Mos. Change
Undifferentiated	34	+7.8	29	+ 8.9
Hypothyroid	4	+7.0	4	+12.8
Schizophrenic	3	+9.3	2	+10.5
Birth Injury	5	+5.4	2	+ 7.7
Post Infectious	1	+7.0	1	+ 6.0
Familial	1	—7.0	1	— 3.0
Normal (I.Q. 95)	3	+9.3	2	+17.0

A word might be said concerning the number of patients classed as mental defectives who are actually childhood schizophrenics, with their bizarre pattern of testing. This type of child, with his lack of rapport, general intractability and inability to fit into even the most favorable home situation, would be likely to be placed in an institution. Likewise the patients with severe degrees of mental damage, whose lack of social adaptation make them a danger or a burden at home and to the community, would find their way into an institution. Thus, there is little common ground for comparison of the institutional studies with the studies conducted in out-patient situations.

A major difference between the present study and those of Loeb, McCulloch, and Fuller, is their use of monosodium glutamate instead of glutamic acid as the therapeutic agent. The efficacy of the salt has been compared to the free glutamic acid by Waelsch,[10] who found that children who had previously responded to treatment with glutamic acid therapy did not show any improvement when potassium glutamate was substituted in amounts equivalent to their previous dosage. Further evidence is furnished by the studies of Pond[17] which show that twenty times as much free glutamic acid is excreted in the urine after a test dose of monosodium glutamate than after an equivalent dose of glutamic acid. Since the amount of free glutamic acid in the urine is only a small fraction of the total glutamic acid excreted normally, extension of this work to cover the total excretion of glutamic acid in urine after test doses of the neutralized and free acids must be done before it can be accepted that the difference between the clinical effect of glutamic acid and of monosodium glutamate is based on the more rapid excretion (loss) of the sodium salt. This report by Pond also shows that there may be a clinical difference between glutamic acid and its salts in their effect on petit mal epilepsy. The salts increased the spike and dome activity, whereas the free acid did not. In view of the clinical and biochemical evidence to date, it is impossible to compare changes in I.Q. on treatment with glutamic acid with clinical experiments with monosodium glutamate.[18]

The ceiling effect predicted by Zimmermann,[12] seems to have operated to some extent in our group, since there was diminishing improvement, progressively over two years of treatment. (Table 2.) Even after two years, however, there was still an increasing mean I.Q. suggesting that observations over a much longer period will be necessary to make any definite statements about effective duration of therapy. Since improvement in many of our cases did not begin until the second 6 months on glutamic acid, our findings suggest that at least a full year of therapy is necessary before it can be deemed ineffective.

A theoretical basis for the use of glutamic acid is fairly well established with more recent evidence suggesting a significant role of glutamic acid in carbohydrate metabolism of the brain.[19,20,21] The positive results obtained in patients in environments conducive to mental improvement suggest that this subject be studied further with larger groups of patients for even longer periods of time. These results also suggest that although large changes in mental status are not to be expected, nevertheless there is reason to believe that a chemical lesion, involving glutamic acid metabolism is present in some mental defectives. We are trying to uncover some criterion by which this type of patient can be detected other than by a clinical trial. This study encourages us to investigate analogues of glutamic acid which are known to be involved in cerebral metabolism.

Summary

1. Fifty-one patients of I.Q. range 37 to 99 were studied for their mental growth response to treatment with glutamic acid.

2. Data on mental growth for 15 of these patients were obtained for periods ranging up to 8 years, and a mental growth curve was plotted. The mental growth of these same children was then plotted during treatment with glutamic acid, and a significant change in rate of mental growth was found.

3. For the entire group of 51 patients, the mean increase in I.Q. over the first 6 months of treatment with glutamic acid was 7.37 points; over 12 months 9.02 points; over 18 months 11.32 points; and over 24 months 13.77 points. These results are all significant with a probability of less than 1 part in 1,000.

4. On discontinuing glutamic acid therapy, with a change to placebo or no therapy, there was a mean drop in I.Q. from the previous test of 6.68 points.

5. Comparison of the results of other investigators indicates that the patients who live in home situations, or situations conducive to learning, show some improvement on glutamic acid, whereas institutionalized mental defectives show no improvement.

Authors' Note: This study was made possible through grants from the International Minerals and Chemical Corporation and from the Child Welfare Society of Children's Hospital. L+ Glutamic Acid was supplied by the International Minerals and Chemical Corporation, Chicago, Illinois.

A preliminary report of these data was presented in the form of an exhibit at the Annual Meeting of the American Academy of Pediatrics in Toronto, Canada, October 1951.

We wish to thank Dr. S. P. Bessman, presently Director of the Glutamic Acid Research Project, for aid in the preparation of this manuscript. The authors take this opportunity to express their gratitude to Dr. Bernard Halperin of the National Institutes of Health for advice in respect to the statistical analysis, and to Miss Carolyn Sweeney, Mrs. Mary Hoover, Mrs. Nora Gross, and Mrs. Mary Lou Hobbs.

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A Neurotic Index

(A Numerical Scoring System for Evaluating Neurotic Patients)

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An index in the form of a chart is proposed as an efficient and comprehensive means of evaluating the course of therapy on neurotic patients. My associates and myself have used this Neurotic Index in connection with our carbon dioxide treatments and found that the chart supplied accurate and detailed information which might otherwise be unavailable. With this chart tabulation is easy and rapid.

It has always been a matter of much difficulty to measure symptoms and emotions in regard to intensity and frequency with any degree of exactitude. Such a measure in some sort of standard form is vital in order to make an evaluation of patients' symptoms and reactions to treatment and to combine groups for statistical compilation. Also, it is desirable to compare results obtained with different forms of treatment. All sciences use mensurable methods and some scientists go so far as to say that all science is fundamentally a form of mensuration. Unless we adopt a yard stick for evaluating instead of pure impressionism we will not have a true scientific basis for psychiatry. Tests such as the Rorschach and numerous others can be reduced to figures but they are usually difficult to understand and estimate. An attempt is being made here with this Neurotic Index to simplify the entire procedure and reduce it to such form that it can be easily read, understood and used by any clinician.

There are 54 major classifications arranged alphabetically in this form. This is acknowledged to be a crude attempt to summarize all the neurotic symptoms but it is the best I have been able to devise up to now. One of the classifications, number 23, homosexuality, is debatable as a neurotic symptom but it is sufficiently frequent in certain of its forms to either cause severe anxiety or as a reaction to neurotic states to merit inclusion. Number 54 covers any symptom not listed.

The top of the form carries the usual information as to the patient's name, age, occupation, and a summary of the clinical condition and diagnosis. The exact type of treatment is noted and the first evaluation should be made at the time the patient is originally seen before any treatment is given.

In Figure 1, an example of the Neurotic Index as filled in, there are three groups of three columns each. Each group is for a separate evaluation. The first column of each group is marked F, the second I and the third P. Under these there are numbers following each symptom. The explanation for these numbers is found in a footnote. Frequency (F) is noted in a range 0, 1, 2, 3 and 4, representing none, infrequently, one

JACKMAN NEUROTIC INDEX

NAME John Doe NO. 64 SEX Male AGE 28

SUMMARY AND DIAGNOSIS: Anxiety reaction, severe, chronic, of 10 years duration, getting progressively worse.

TREATMENT: CO_2 inhalation, 30% CO_2 70% O_2, multiple breaths.

SYMPTOMS	CLINICAL EVALUATION								
	Pre Rx Date			*Rx No. 30 Date*			*Rx No. Date*		
	F*	I**	P***	F	I	P	F	I	P
1. Anxiety	4	3	12	1	1	1			
2. Alcoholism									
3. Allergy									
4. Anorexia	3	2	6	0		0			
5. Asthma or shortness of breath									
6. Compulsion									
7. Constipation									
8. Concentration difficulties									
9. Cephalgia									
10. Cardiac disturbances									
11. Depression	3	3	9	1	1	1			
12. Dependency									
13. Diarrhea									
14. Dizziness and lightheadedness									
15. Drug addiction									
16. Enuresis									
17. Employment difficulties	4	3	12	0		0			
18. Fatigue									
19. Frigidity									
20. Family relationship difficulties									
21. Guilt feelings									
22. General aches and pains									
23. Homosexuality									
24. Hypochondriasis									
25. Impotence	2	3	6	0		0			
26. Indecision									
27. Indigestion	2	2	4	0		0			
28. Inferiority									
29. Insecurity	3	2	6	1	1	1			
30. Irritability	4	3	12	1	1	1			
31. Impulsive tendencies									
32. Involuntary movements									
33. Inhibited									
34. Insomnia									
35. Instability (emotional)									
36. Masturbation									
37. Migraine									
38. Menstrual difficulties									
39. Nail biting									
40. Nausea and vomiting									
41. Obsession									
42. Paralysis									
43. Phobia									
44. Self-consciousness									
45. Skin disturbances									
46. Spastic colon	2	1	2	0		0			
47. Stuttering									
48. Social participation difficulties									
49. Tension									
50. Ulcer									
51. Vasomotor disturbances									
52. Weakness									
53. Worrying	4	3	12	0		0			
54. Other symptoms									
NEUROTIC INDEX			81			4			

*FREQUENCY

0—None
1—Infrequent
2—About half the time
3—Most of the time
4—Always

**INTENSITY

1—Minimal (present to minor degree)
2—Moderate (halfway between minimal to severe)
3—Severe (greatest intensity possible for individual and situation)

***Product is result of multiplying frequency by intensity
NEUROTIC INDEX is sum of P's.

half the time, most of the time, and continuous. Intensity (I) is signified by the numbers 1, 2 and 3, representing minimal (present to minor degree); moderate (halfway between minimal and severe); and severe (greatest intensity possible for individual and situation). Obviously this is expressing impressions in figures but by reducing impressions to numbers we have a very effective, simple and logical handle to grasp instead of having to go into a long, confused and cumbersome story as to how much and how strong the patient's symptoms are. In addition we can compare the various stages that the patient goes through in responding or not responding to treatment. In our example, anxiety which is present in any neurosis is present continuosly and in a severe state. Therefore, in the first column F after anxiety we would put down 4, and in the second column I, we would put down 3. We would go down the list with the patient checking all 54 possible symptoms and at line 54 include anything that may not be included specifically. Particular symptoms can be described in greater detail by a qualifying word in the space following the symptom.

The third column P is the product of F and I. Thus we can reduce the intensity and frequency of each symptom to one number. At the bottom of the P column all the numbers are totaled to give a single sum which is called the Neurotic Index. This single number gives you a numerical picture of the patient's clinical state before treatment. After the patient has received 30 treatments, or it is decided to discontinue treatment, or we decide to evaluate the results at any point, we can repeat the procedure of evaluation. In our example in Figure 1 the neurotic index before treatment was 81 and after 30 treatments it was 4. This gives a direct numerical comparison or index as to the difference between the patient's condition before and after treatment.

In CO_2 therapy a re-evaluation is helpful after 30 treatments. A large number of cases can be quickly summarized numerically and the result of numerous clinical investigations indicated quickly and the results of various forms of treatment can be compared.

There are many questions that would arise in using this Neurotic Index chart. For example, the question as to why we multiply F by I to get P. The answer is that they are multiplied to get an area picture which gives a profile of each patient's symptoms similar to that on a graph. Adding the numbers does not disclose any pertinent information, does not disclose the profile and beclouds the issue.

If a patient had severe attacks infrequently before treatment and following treatment had minimal attacks most of the time, multiplying the corresponding numbers would still give you the same number, 3. This is not an error, but actually is a good picture because now the severe attacks are less intense though more frequent. The patient is still suffering collectively just as much as he did before. So the number 3 for the product before and after treatment would be valid as essentially there was no real change. However, if one wishes to qualify that figure one can add on the back of the chart a qualifying or explanatory statement which can be mentioned in the complete summary of the individual or of the series treated.

As far as evaluation of intensity and frequency is concerned, this evaluation is made by the patient and is subjective and is elicited by the psychiatrist from the patient. After all, when the patient first appears we can do no more than accept his own evaluation of his condition. A similar possible degree of error will enter into the picture when the patient evaluates himself the second or third time. I do not consider this a defect; on the contrary, if the patient evaluates his symptoms before the treatment and is very disturbed by it, thus causing some "error" in his evaluation, the fact that there is decreased emotional reaction in his evaluation when he is improved at the end of treatment would be a further indication that the treatment has been effective and it would show in his evaluation. Of course, the psychiatrist in noting his evaluation would act as an objective brake and help stabilize the patient's emotional over-reaction in estimating intensity and frequency.

It is hoped that this method may serve the purposes mentioned and serve as a stimulus to better methods of mensuration in psychiatry.

A Clinical Report on the Palm-chin Reflex

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The purpose of this study is to report our experiences in routine examinations of 800 patients in a diagnostic center and the admission service of a state hospital on the value of the palm-chin reflex, sometimes referred to as the "palmo-mental reflex."

During the past few years increasing attention has been focused on the significance of this reflex, which was first described by Marinesco and Radovici in 1920. It consists of an ipsilateral contraction of the mentalis on painful stimulation of the thenar eminence. Occasionally the mentalis response is bilateral or involves the obicularis oris. This reflex is best elicited by having the patient extend the hand somewhat and, while observing the mental area, briskly stroke the thenar eminence with a key, tongue blade, or some similar object. The positive response is considered to be present if there is a contraction of the ipsilateral mentalis or obicularis oris. In many cases this response is barely perceptible and easily exhausted. The authors consider a response to be positive only if it is moderate to marked and capable of being repeated immediately. Most of the positive responses were found to be exhaustible with numerous repeated stimulation.

There has been much debate over the validity of the palm-chin reflex. The following table reveals the discrepancy in findings on normal subjects by various investigators:

TABLE I

	No.	*Incidence*
Marinesco and Radovici	300	50%
Sarno	100	8%
Diaz	208	11%
Thompson	Many	2%-3%
Blake & Kunkle	170	49%
Burton & Miller	200	2.5%

In view of this marked variation in the frequency of positive responses in normals, we will define "normal" as used in our study as:

A person under 50 years of age with absence of findings or complaints referable to the central nervous system and absence of findings of hypertensive cardiovascular disease or arteriosclerotic changes.

We do not know what was regarded "normal" by the other authors. Our material was drawn from the Manteno State Hospital and the Behavior Clinic of the Criminal Court of Cook County. Our normals were employees in Manteno State Hospital and inmates in the Cook County Jail who were referred by court order for examination by the Behavior Clinic and who fit into our category of normals. Of the so-called normals tested we found the reflex present in 3 out of 100, or 3%. A detailed neurological examination on the 3 normals who showed a positive palm-chin response, failed to reveal any evidence of central nervous system disease. Their ages were 27, 42 and 48. No correlation or evaluation of personality structure was attempted in the normal group.

Of the 800 patients examined we found a positive response in 180. The palm-chin was performed as part of a routine neurological examination on the admission service at Manteno State Hospital, and on cases referred to the Behavior Clinic of the Criminal Court of Cook County. Additional testing was done on post electro-shock patients and on patients during insulin coma. Table II breaks down those showing positive responses into diagnostic categories:

TABLE II

	No.	Positive	Per Cent Positive
Generalized Cerebral Arteriosclerosis	40	32	80
Amytrophic Lateral Sclerosis	4	4	100
Parkinsonism	36	28	77
General Paresis	13	12	92
Space Occupying Lesions	6	4	66.3
Hemiplegia	23	21	90
Senile Dementia	16	11	69
Depression	38	8	21

From the Behavior Clinic of the Criminal Court of Cook County, Illinois, and Manteno State Hospital.

Schizophrenia	140	3	4.2
Multiple Sclerosis	7	5	76.8
Recovering Acute Alcoholism	70	48	60.8
Recovering Barbiturate Intoxication	4	4	100
Heroin Withdrawal	7	1	14.2
Bell's Palsy	5	0	0
Epilepsy	23	5	21.1

To enlarge on, and further explain, the diagnostic breakdown in Table II, we would like to include the following notes:

Of the 36 cases of Parkinsonism examined, 24 were diagnosed as arteriosclerotic basal ganglia degeneration. Of these 24 cases, 22 showed a positive palm-chin response.

Six space occupying lesions were seen, consisting of 2 subdural hematomas, 1 meningioma, 2 astrocytomas, and 1 papiloma of the choroid plexus with metastatic implants along the aqueduct, producing some internal hydrocephalus. The 2 subdurals and the 1 meningioma were diagnosed by surgical exploration and the 2 astrocytomas and 1 papiloma were observed at post mortem examinations. The subdurals and infiltrating astrocytomas showed a positive palm-chin response, while the papiloma of the choroid plexus and the meningioma failed to show a positive palm-chin. Of the positive palm-chin responses in this group, all 4 were bilateral and, therefore, were of no value in lateralizing the lesion.

We examined 23 hemiplegias, most of whom showed evidence of psychoses indicative of diffuse cerebral involvement. Of these, 16 followed a thrombosis, 1 an embolus and 6 were due to intercerebral hemorrhage. A positive palm-chin was shown in 21 of these cases. However, in 17 the response was bilateral and again of little value in lateralizing the lesion.

Of the 38 persons examined who were classified psychiatrically as depressions, 8 showed a positive palm-chin. Of these 4 were beyond 60 years of age and were considered from physical and fundiscopic findings to have arteriosclerotic changes present. Two others were moderately agitated, and in poor state of nutrition. Following a course of electro-shock therapy the responses in these 2 agitated cases became negative. The other 2 depressions who showed a positive palm-chin were not observed long enough to determine the presence or absence of an organic component. Their initial physical and neurological examinations failed to reveal any evidence of organicity.

In 140 schizophrenics tested, only 3 showed a positive palm-chin. These were all under 50 years of age and the 3 positive were agitated at the time of examination. Here it should be noted that following hyperventilation to the point of vertigo, 2 of 7 normals tested showed a positive palm-chin. This raises the question of whether the positive palm-chin responses were due to excitement and hyperventilation in the schizophrenic group.

It is extremely interesting to note the high percentage of positive responses in recovering alcoholics; during the "rocky-shake" stage of withdrawal 60.8% showed a positive palm-chin. This response persisted for an average of five days following withdrawal of alcohol. During this period many responses became negative if the administration of routine alcoholic care was stepped up. Our routine consisted of elixir of chlorol, paraldehyde, tolserol and short-acting barbiturates. However, when the sedative level was decreased the positive palm-chin responses would return. In 5 cases, 3 of whom were diagnosed as Korsakoff's syndrome and 2 with severe deterioration, a positive palm-chin response persisted for as long as they were observed (3 months). Persons recovering from barbiturate intoxication or a combination of barbiturate and alcohol showed a similar pattern of response. Only 1 of 7 patients recovering from heroin addiction showed a positive response.

As might be expected from the pathology of a peripheral 7th nerve lesion, a positive palm-chin was not elicited in the 5 cases of Bell's palsy observed. We feel the palm-chin is of value in differentiating supra nuclear lesions from nuclear or infra-nuclear 7th nerve lesions.

In our series we had 23 epileptics but only 5 demonstrated a positive palm-chin. Of these, 2 were post traumatic and 3 were idiopathic epilepsy with deterioration. We do not have the diagnostic breakdowns on the other 18 epileptics in this group.

The remainder of our group was made up of behavior disorders, neurotics, personality

dysfunctions and those without evidence of neurologic or psychiatric disease.

Additional testing was done on post-electroshock patients who sometime during the recovery period showed a positive response in 20% of cases. Seven of 10 cases who received electroshock twice daily for several days showed a sustained palm-chin reflex after four or five days of treatment. Insulin therapy at a sub-coma or coma level showed about 70% positive responses. These responses persisted for a short period (5 to 10 minutes after regaining consciousness) following either tube feeding or intravenous termination of the coma. During deep coma the responses could not be elicited in any of the patients tested.

The Nature of the Reflex

The palm-chin reflex is poorly understood. Several factors that may contribute to a better understanding of the mechanisms are as follows:

1. The high incidence of positive responses in diseases of the pyramidal tract.
2. A high incidence in conditions that produce diffuse dysfunction of cortical activities, such as paresis, cerebral arteriosclerosis and senile deterioration.
3. A relatively high incidence in the recovery period of alcoholic intoxication, and a very high incidence following barbiturate intoxication.
4. The presence of the reflex in the post electroshock therapy period, and a relatively higher incidence during insulin sub-coma or coma.

Clinical Value

As noted in Table I, there is little or no agreement on the incidence of the reflex in the normal adult population. Our findings of moderate reaction which can be repeated occurred in only 3% of the normal population that we tested. The presence of the rather high incidence in pyramidal tract disease, diffuse cerebral disease and in cases following toxic suppression of higher center activities (post-alcoholic or barbiturate intoxication) indicates that a positive response should alert one to any of the above conditions.

Our experience leads us to believe that the palm-chin reflex is a simple, rapid means of augmenting a routine neurological examination. The presence of a positive response in a person below 50 years of age indicates that neurologic disease must be ruled out. It must be kept in mind that recent alcohol excesses or barbiturate excesses may alter the patients' responses. The palm-chin reflex value is questionable in more advanced age groups, cardiovascular or arteriosclerotic diseases.

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INDEX 1953 — Volume XIV

Diseases of the Nervous System

ANEURYSM, MYCOTIC ..... September, Pg. 282
Neurological Clinical Pathological Conference, *Charles D. Aring,* Editor
ANXIETY—MEANING OF ..... April, Pg. 110
Edward Podolsky
ARCTIC SURVIVAL RATION: PSYCHIATRIC OBSERVATIONS ..... March, Pg. 83
Joseph J. Reidy
CARBON DIOXIDE CONVULSIVE TREATMENT ..... June, Pg. 179
A. I. Jackman
CARBON DIOXIDE INHALATION THERAPY ..... July, Pg. 209
Albert A. LaVerne and Morris Herman
CARBON DIOXIDE INHALATION THERAPY—RAPID COMA TECHNIQUE ..... May, Pg. 141
Albert A. LaVerne
CARBON DIOXIDE INHALATION THERAPY—VARIATIONS ..... September, Pg. 269
Albert A. LaVerne
CARBON DIOXIDE MAINTENANCE THERAPY IN NEUROSES AND ALCOHOLISM October, Pg. 316
Albert A. LaVerne and Morris Herman
CARBON DIOXIDE TREATMENT—CINEMATOGRAPHIC ANALYSIS OF REACTIONS July, Pg. 212
L. Douglas MacRae
CARBON DIOXIDE—TREATMENT OF STUTTERERS WITH ..... August, Pg. 243
Anna May Smith
CARBON DIOXIDE TREATMENT USING NITROUS OXIDE ..... April, Pg. 102
L. J. Meduna
CARBON DIOXIDE TREATMENT—STANDARDIZATION ..... August, Pg. 240
Robert E. Peck
CARBON MONOXIDE POISONING ..... February, Pg. 39
E. Adams Daneman
CEREBRAL ARTERIOSCLEROSIS ..... July, Pg. 215
Neurological Clinical Pathological Conference, *Charles D. Aring,* Editor
CEREBRAL TRAUMATA—VALUATION OF PSYCHOPHYSIOLOGIC AFTERMATHS ..... June, Pg. 163
Lewis J. Siegal
CONGENITAL ABSENCE OF THE ODONTOID PROCESS ..... August, Pg. 236
Naomi Raskin
DIAGNOSIS, PATHOGENESIS AND TREATMENT OF MENTAL DISORDERS ..... July, Pg. 204
Solomon Katzenelbogen
DORMIN POISONING ..... August, Pg. 239
G. Donald Niswander
DORMISON AS AN ADJUVANT IN ELECTROCOMA THERAPY ..... June, Pg. 172
W. C. Brinegar and A. Sainz
E.E.G. PATTERN VARIATION IN NEUROSYPHILIS ..... May, Pg. 138
Leslie J. Seeley and Rodgers S. Dille
ELECTRIC SHOCK IN PSYCHOSIS WITH CEREBRAL SPASTIC PARALYSIS ..... April, Pg. 99
Paul Lowinger and Paul E. Huston
ELECTRIC SHOCK THERAPY OBSERVATIONS ..... February, Pg. 35
W. E. Wilkinson
ELECTROENCEPHALOGRAM IN POST-TRAUMATIC HEADACHE SYNDROME ..... November, Pg. 335
Israel H. Schuleman
ELECTROENCEPHALOGRAPHIC SURVEY OF A MENTAL HOSPITAL ..... November, Pg. 331
Eugene Revitch and Michael Luzzi
ELECTROSHOCK THERAPY—ASPIRATION IN ..... November, Pg. 328
Ernest Kahn
EMBOLISM, FAT ..... January, Pg. 24
Neurological Clinical Pathological Conference, *Charles D. Aring,* Editor
EMPYEMA, SUBDURAL ..... April, Pg. 112
Neurological Clinical Pathological Conference, *Charles D. Aring,* Editor
ENCEPHALOGRAMS—REVIEW OVER 5 YEAR PERIOD ..... December, Pg. 355
Israel H. Schuleman
FRONTAL LOBES ..... March, Pg. 73
Frank R. Ervin
GLUTAMIC ACID (L+)—EFFECT ON MENTAL GROWTH ..... December, Pg. 368
Dorothy S. Jaeger-Lee, Emma Gilbert, John A. Washington, and Jonathan M. Williams
GROUP PSYCHOTHERAPY REACTIONS OF PATIENTS ..... May, Pg. 144
J. W. Klapman
GROUP PSYCHOTHERAPY WITH WAYWARD GIRLS ..... October, Pg. 308
Benjamin Kotkov
HEPATOLENTICULAR DEGENERATION—ADVANCES IN THERAPY ..... March, Pg. 95
Irvin M. Cohen
HISTAMINE—ACUTE EFFECTS IN PSYCHIATRIC PATIENTS ..... September, Pg. 259
John W. Lovett Doust
HOMO-ANONYMOUS ..... October, Pg. 318
Ernest Harms

HOW LONG WAS ROOSEVELT ILL BEFORE HIS DEATH? ..... November, Pg. 323
W. G. Eliasberg
HYPNOTHERAPEUTIC FIRST-AID IN MILITARY SERVICE ..... February, Pg. 47
Jerome M. Schneck
HYPNOTHERAPY FOR THE CONTROL OF ANXIETY ..... September, Pg. 274
Jerome M. Schneck
HYSTERICAL AMNESIA TREATED BY HYPNOSIS ..... October, Pg. 313
Sanford M. Izner, John Goldman and Rudolf Leiser
INTEGRATION OF THE DISINTEGRATED PERSONALITY ..... October, Pg. 305
James L. McCartney
INVERT SUGAR IN INSULIN COMA THERAPY ..... October, Pg. 291
Morriss Mills, Jr. and Floy J. Moore
ISONICOTINIC ACID HYDRAZIDE IN NONTUBERCULOUS MENTAL PATIENTS .. September, Pg. 278
W. P. Wilson
LOBOTOMY RESULTS ON SEVENTEEN STATE HOSPITAL PATIENTS ..... June, Pg. 176
Grady Niblo
MANAGEMENT OF DISTURBED PATIENT BY SEDATIVE ELECTROSHOCK ..... January, Pg. 21
Lewis I. Sharp, Anthony R. Gabriel and David J. Impastato
MATRICIDE WITH EVIDENCE OF THALAMIC OR HYPOTHALAMIC DISORDER ..... January, Pg. 18
Edward D. Schwade and Sara G. Geiger
MULTIPLE SCLEROSIS ..... May, Pg. 152
Neurological Clinical Pathological Conference, *Charles D. Aring*, Editor
MUSCULAR DYSTROPHY, LATE ..... June, Pg. 183
Neurological Clinical Pathological Conference, *Charles D. Aring*, Editor
NARCOSYNTHESIS EFFECTS OF SODIUM AMYTAL, METHEDRINE AND L.S.D.-25 .... March, Pg. 85
Solomon Katzenelbogen and Ai Ding Fang
NEUROGENIC ULCER OF DUODENUM ..... February, Pg. 55
Neurological Clinical Pathological Conference, *Charles D. Aring*, Editor
NEUROTIC INDEX ..... December, Pg. 375
A. I. Jackman
NEUROTIC SYMPTOM OF "DRIPPING HANDS" ..... July, Pg. 202
Edmund Bergler
NUVARONE IN THE TREATMENT OF EPILEPSY ..... April, Pg. 108
John H. Taterka and Charles B. Thompson
NEUROSYPHILIS, ASYMPTOMATIC ..... August, Pg. 245
Neurological Clinical Pathological Conference, *Charles D. Aring*, Editor
PALM-CHIN REFLEX—A CLINICAL REPORT ..... December, Pg. 378
John J. McLaughlin and William H. Haines
PALSY, SUPRANUCLEAR ..... February, Pg. 55
Neurological Clinical Pathological Conference, *Charles D. Aring*, Editor
PARAPLEGIA CONFERENCE ..... November, Pg. 342
Neurological Clinical Pathological Conference, *Charles D. Aring*, Editor
POLIOMYELITIS IN CALIFORNIA ..... December, Pg. 366
Page Stelle
PSYCHOPATHIC PERSONALITY—SOCIAL IMPLICATIONS ..... October, Pg. 296
Edward E. Mueller and Burman H. Preston
PITRESSIN—EFFECT ON BLOOD PRESSURE IN PSYCHOTIC PATIENTS ..... September, Pg. 279
D. E. Macdonald
PITRESSIN IN SCHIZOPHRENIA—COMPLICATIONS ..... May, Pg. 149
Lorant Forizs and Burton L. Travis
PROGRESS IN HOSPITAL TREATMENT ..... May, Pg. 131
Orkar Diethelm
PSYCHIATRY IN CENTRAL EUROPE ..... August, Pg. 227
Otto Schwarz
PSYCHIC MASOCHISM—UNDERSTANDING OF ..... November, Pg. 340
Edmund Bergler
PSYCHOMOTOR EPILEPSY IN CHILDREN ..... November, Pg. 337
Richard C. Robertiello
PSYCHOPATHIC DELINQUENTS ..... February, Pg. 52
P. R. Newkirk
SIBLING RIVALRY ..... March, Pg. 67
Raymond W. Waggoner
SODIUM AMYTAL AS AN AID IN PSYCHOTHERAPY ..... January, Pg. 14
Milton H. Rodofsky
SODIUM AMYTAL NARCOSIS, WITH DELIRIUM, AS A THERAPEUTIC METHOD ..... July, Pg. 195
Eugene J. Alexander
SUBDURAL HEMATOMA ..... August, Pg. 245
Neurological Clinical Pathological Conference, *Charles D. Aring*, Editor
THROMBOSIS OF SUPERIOR LONGITUDINAL SINUS ..... March, Pg. 89
Neurological Clinical Pathological Conference, *Charles D. Aring*, Editor
THYROID THERAPY IN MENTAL DISORDERS ..... January, Pg. 3
Lewis Danziger and Josef A. Kindwall

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